An Address
ON
CHRONIC INTESTINAL STASIS:
"AUTOINTOXICATION" AND SUB-INFECTION.

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STEADILY and progressively during the last score or so of years there has developed a tendency to ascribe to derangement of the intestinal tract the origin of not a few grave disorders, until to-day we have Sir Arbuthnot Lane[1] recording no fewer than seventeen more or less outstanding symptoms as directly due to stasis and delayed passage of faecal matter through the lower end of the ileum and the larger bowel, together with nine maladies indirectly due to the same cause, while we are informed that these are merely the more important possible outcomes of stasis; several others may also be imputed to this cause. It is a portentous roll—a roll so long that we may well call a halt and examine into its details.

SIR ARBUTHNOT LANE'S THESIS.

Let me, as this is to form the text for my sermon, rapidly recapitulate Lane's conclusions:

By chronic intestinal stasis, says Sir Arbuthnot, he means that the passage of the contents of the intestinal canal is delayed sufficiently long to result in the production, in the small intestine especially, of an excess of toxic material, and in the absorption into the circulation of a greater quantity of poisonous products than the organs which convert and excrete them are able to deal with. In consequence there exist in the circulation materials which produce degenerative changes in every single tissue of the body, and lower its resisting power to invasion by delcterious organisms.

He ascribes these disturbances primarily to delay of faecal material in the large bowel. This loading of the large bowel tends towards downward displacement and dragging upon the other organs in the abdominal cavity. Along the lines of stress there develop definite membranes; these very membranes, by pulling upon the other organs, induce kinks and partial obstructions, and where this obstruction tells upon the small bowel dilatation occurs above the point of obstruction, with a relative stasis. Thus we find brought about a delayed passage of the contents of the small intestine, as also of the duodenum and the stomach, with resultant infection of these organs and accumulation of deleterious bodies from the stagnating material. He enumerates the following as the symptoms which result directly from the "autointoxication" of chronic intestinal stasis:

1. Loss of fat.
2. Wasting of both voluntary and involuntary muscles.
3. Alteration in the texture and colour of the skin, with pigmentation and offensive perspiration.
4. Subnormal temperature, especially affecting the extremities. There is no abrupt line of separation between this condition and Raynaud's disease, of which it would appear to be a stage.
5. Mental conditions of apathy, stupidity, or misery, which may become exaggerated to a state of melancholia, or even apparent imbecility, with suicidal tendencies. There may be neuralgic symptoms, neuritis, frequent headache, loss of control over the temper. These nervous states due to stasis are a much more frequent cause of serious crime than is generally imagined.
6. Rheumatic aches and pains in muscles, joints, and skin.
7. Atrophy of the thyroid gland.
8. Either increased or lowered blood pressure,
9. Degenerative changes in the breasts, especially in the upper and outer zone of the left breast, predisposing to cancer.
10. Prolapse of abdominal organs, partly because of loss of fat, partly because of wasted muscle fibres. Increased mobility of the kidneys, and prolapse and bends of the uterus.
11. Breathlessness on exertion, at times of asthmatic type, due in some cases to a distension of the stomach and intestines.
12. Degeneration of the heart muscle with dilatation of left heart and aorta and arterio-sclerotic changes (atheromatous) in the systemic arteries.
13. Renal changes which are roughly grouped under the term "Bright's disease."
14. Early loss of hair colour with falling out, more common in those with dark than with red hair.
15. Affections of the pancreas with chronic induration, inflammation, and finally cancer. Pancreatic diabetes.
16. Infection of the biliary system, cholecystitis, cholelithiasis, cancer, together with many acute and chronic diseases of the liver.
17. Degenerative diseases of the eye.

The indirect changes resulting from the lowered resisting power of the tissues can, says Sir Arbuthnot, hardly be separated abruptly from these more direct changes. Of these the most obvious are:

1. Infection of the gums and pyorrhoea alveolaris.
2. Tuberculous infection other than that due to direct inoculation, as through the skin.
3. Rheumatoid arthritis. This, like tuberculosis, is declared not to develop, except in the presence of defective drainage of the gastro-intestinal tract.
4. Infection of the genito-urinary tract, producing nephritis, cystitis, pyelitis, endometritis, salpingitis, etc.
5. Thyroid disorders, such as diffuse enlargement, exophthalmic goitre, adenomatous tumours.
6. Still's disease.
7. Pustular infections of the skin.
8. Infections of the large intestine; several varieties of mucous and ulcerative colitis.
9. Ulcerative endocarditis.

These, says Sir Arbuthnot, are merely a few obvious, typical, indirect results of the "autointoxications" of chronic intestinal stasis.

On the occasion when he announced these conclusions Sir Arbuthnot brought forward seventeen patients and their case reports as evidence that these various conditions can be cured or definitely ameliorated by short-circuiting the large intestine, by the operation of ileo-sigmoidostomy with or without removal of the colon, by removal of obstructing bands, or, again, by performance of gastro-jejunostomy to overcome duodenal obstruction.

To-day I want to consider, not as a clinician, but as a pathologist, how far we may reasonably accompany Sir Arbuthnot, to what extent his doctrine is to be accepted; for, honestly, at first sight these seventeen symptoms, and nine diseases indirectly induced, seem to be a horrible jumble. Instinctively we are thrown into an attitude of doubt; instinctively we feel that the great surgeon of Guy's Hospital, to whom surgery owes so much, may have fallen a victim to that most dangerous, if it be not the last, infirmity of noble minds—obsession. For it seems to me that the noble mind as distinct from the great mind possesses a certain quality of generous enthusiasm which, while it is an added virtue, is also a danger; so that its possessor, once he has discovered a truth, in that enthusiasm inevitably sees applications of that truth in all directions. If it so happens that he has lighted upon a fundamental truth, this is a perfectly safe procedure; we marvel at his perspicacity, and the charge of obsession, if advanced, passes off like water from a duck's back. If his discovery is not a fundamental truth, but only a half-truth, then the same obsession leads him with fatal sureness to some *reductio ad absurdum;* leads him to enumerate as related—and what is more, to be convinced that they are related—matters which the every-day man not thus wrought up recognizes as capable of a totally different and more natural explanation. And the pitiful part of it all is that small minds and untrained—as we see every day in matters of religion—may be possessed of a like enthusiasm over what are not even half-truths, but merely phantasms of a disordered imagination; and a callous world, heedless of distinctions, judges both orders of men by the same judgement, and cheerfully consigns both to the common pit.

I speak here with all sympathy, for I have been there. I have known what it is in the course of my work to have reveal itself to me what appeared to be a great principle, to find that principle applying itself to and elucidating every phase of my subject, until all my thoughts throughout the day, and in the watches of the night, hovered around and constantly returned to this one principle and its application, to the virtual exclusion of everything else; until, frankly, I became frightened at myself, and with a determined effort gave up that line of investigation—turning to routine university work. I had known others—

workers of higher calibre than myself—who had become similarly so filled with their work and its possibilities that temporarily, it seemed to me, their reason had become somewhat unbalanced. And here is the point: it was about this very matter of intestinal derangements that I thought I saw light. To me also it appeared that they were the cause of many grave conditions—in fact of so many and so diverse that, as I say, I feared to continue further, lest if I were not obsessed the world nevertheless would so regard me. That was fourteen years ago, and in the meantime I have largely permitted this particular field to lie fallow. Taking these facts into consideration, I think it will be admitted that I can deal with this subject both sympathetically and—shall I say?—with a certain maturity of judgement.

Is Sir Arbuthnot Lane justified in his teaching, or is he the victim of obsession?

"AUTOINTOXICATION."

For practical purposes the present movement began with Professor Bouchard,[2] and with a blunder which has shown its fell influence all through these years up to the present moment. The blunder is all the more remarkable as having been perpetrated by a Frenchman, for to the leaders of French thought, be it in literature, in the sciences, or in medicine—and Bouchard was such a leader—we look for clear, logical exposition of their subjects, and particularly for a choice and precise use of words. It was in 1887 that Bouchard published his lectures upon "autointoxication" in disease; to him we owe the popularization, if not the actual introduction, of this miserable word—a word, let me say, that is not miserable in itself, but deserving of condign damnation when wrongly applied, as Bouchard employed it, as a means to cloak our ignorance in a garment of pretended knowledge. For in a most muddleheaded manner, among the self-poisonings proper — the poisonings, that is, due to excess or defect of the products of metabolism, to disordered working of the body cells themselves, or due, again, to the actual disintegration of cells and the products of dissociation of dead tissue—Bouchard not merely included, but laid particular emphasis upon, a group of intoxications which by no stretch of the imagination can justifiably be termed self-poisonings. The poisons produced by invading bacteria are of extrinsic origin; of course they are not autointoxicants of the body. It is absurd to retort that what Bouchard meant is that when they are produced in our "insides" they become part of ourselves, and so auto-intoxicants. As I pointed out many years ago,[3] Bouchard had no right to employ any such slipshod convention. For, accurately, the contents of the intestines are not inside—they are outside the body; only that is inside the body which has penetrated through the cellular lining or surface membrane. Poisons, therefore, originating within the alimentary canal, originate outside the body every whit as much as if they had their origin in the vaginal chamber, or on the skin. Even if they were derived from the products of cellular excretion into the alimentary canal—from substances not in themselves poisonous, but rendered poisonous by the dissociative activities of bacterial or other external agents—they still would not be autointoxicants. Still less are the products of foodstuffs, taken from without, autointoxicants, no matter whether these products gain their poisonous properties through the action of the digestive juices or through the putrefactive functions of the intestinal bacteria. Bouchard, however, with sublime heedlessness, included all these as inducing autointoxication; meat poisoning, whether infective or ectotoxic, internal strangulation, and frank infections like typhoid fever and cholera, were by him all comprehended as autointoxications of intestinal origin.

This blessed word "autointoxication," *sensu Bouchard*, has become a shibboleth; and after the inevitable tendency of frail humanity it is scarcely ever employed for the self-poisonings proper; we see it used every day, both by those who ought to know better—like Sir Arbuthnot Lane and Dr. Saundby—and yet more often by those who obviously do not know better, as a blanket or omnibus term to cover all the processes which are not autointoxications, processes whether of toxic or infectious nature originating in the alimentary canal. As a consequence, the users are blinded to the fact that they are confusing together many processes which for the purposes of clear thinking and progress ought to be carefully kept apart.

The word, in short, should and must be banished from the vocabulary of all self-respecting medical men. It is absurd to jumble all these conditions into one common heap; our duty is to recognize and classify each different form—to analyse before we attempt to synthetize.

THE ETIOLOGY OF INTESTINAL STASIS.

Herein, speaking as a pathologist, is my main criticism of Sir Arbuthnot Lane's paper. Let us admit that all these symptoms which he enumerates are found to follow intestinal stasis; let us admit all his list of diseases said to be indirectly due to the same condition. He himself will be the first to admit that in any given case only a relatively small proportion of the symptoms are present; in any given case only one or two of the diseases he enumerates indirectly follow the development of the condition. The variation is too great to be put down to individual idiosyncrasy. There must be different agents at work in different cases. Even if we grant that one and all are relieved by short-circuiting—this does not mean that stasis is the essential predisposing factor—those agents, by analogy, are likely to gain entrance by various tracts. To say, as Lane at least implies, that without intestinal stasis there would be no generalization of tuberculosis, is, it may be urged, to ride one's horse to death.

If words have any weight, then the logical result of his teaching is that, for instance, so soon as we diagnose a case of tuberculosis we are to whip out that patient's colon, because, forsooth, without intestinal stasis we should have no tuberculosis, save that induced by direct inoculation. Here, surely, is the *reductio ad absurdum* of the half-truth. Does the cow with its dozen and more evacuations per diem suffer from intestinal stasis? Does it suffer from displacement of the abdominal viscera due to the assumption of the "all fours" position, and, notwithstanding, is it not quite as susceptible to tuberculosis as is man?

It would have been within the bounds of moderation to call attention to the fact that where stasis exists in certain cases of chronic progressive tuberculosis, the general condition of the patient is so much improved by short circuiting that a definite arrest of the disease occurs. In like manner it is necessary to point out that conditions such as cholelithiasis, cirrhosis of the liver, pancreatic diabetes, are, like tuberculosis, indirectly caused by conditions other than intestinal stasis.

From another point of view the objection may be raised that Sir Arbuthnot Lane, as has been pointed out to me by my colleague, Professor Geddes, has not gone far enough; he has not recognized the *fons et origo mali*. In a long survey of the anatomical relationships of the viscera, Dr. Geddes assures me that constantly where he has encountered Jackson's membrane or Lane's and other bands of like nature, he has found a lax abdominal wall with more or less atrophy of the recti and other muscles. The prime cause of visceral displacement is lack of due support. To remedy this, rather than to give paraffin, should be the first object of preventive medicine and surgery.

As a pathologist two questions present themselves to me. The first: Granting that by bismuth meals or other means we discover that there is serious delay in the passage of the contents of the alimentary canal, and that accompanying this delay a certain number of the symptoms described by Lane have made their appearance, is this sufficient ground for operation? The second: What is the relative value of these various symptoms as an indication for operation? or, put otherwise, do we know anything definite regarding these symptoms and the way in which they are brought about, and can we utilize that knowledge as a guide in determining our mode of treatment?

On "Lane's Bands" as an Indication for Operation.

In answer to the first, I am inclined to say that, provided the fluoroscope demonstrates the existence of kinks and relative obstruction by narrowing of the lumen in any region, operation is to be recommended—operation, that is, to remove the bands causing the kinks or obstructions.

No medical treatment can permanently restore the *status quo ante;* at most, dieting on soft foods and carefully adjusted support of the anterior abdominal wall may palliate the conditions. It is true that, as Sir Arbuthnot admits, without such support after operation these bands are liable to form again, but operation gives a greater chance of removing the obstruction than any other means known to us.

I do not like to seem to be blowing my own trumpet, but where another worker affords illustrations which either support or oppose one's own favourite doctrines, it is difficult to be silent. Thus for long years I have dwelt upon the existence of non-inflammatory fibroses [4] and what I term stress hypertrophy of the connective tissues, pointing out that the effects of strain as distinct from overstrain are increased development of the tissue subjected to the stress. Judged by the little reference to this teaching by other pathologists, I have sometimes thought of myself as one crying in the wilderness. Some may know that it is in this way that I explain the overgrowth of the intima and adventitia of arteries in arterio-sclerotic conditions. Lane likewise feels that he is a *vox clamans in deserto.* I have to confess ignorance of the fact that he as an anatomist and surgeon has been proclaiming this doctrine since 1887.[5] There could be no prettier demonstration of the action of this principle than is afforded by these bands upon which he lays so much emphasis, these evolutionary bands as he terms them, produced by the pull of the badly-supported bowel upon its mesenteric attachments.*

When we remember that the whole process of digestion is one of conversion of the foodstuffs into a soluble fluid state, we may assure ourselves that the absorption of fluid that occurs in the colon is at the same time an absorption of foodstuffs, while the very concentration of this faecal matter arrests bacterial activity in an extraordinary manner. It is a preventive of the putrefactive changes which originate in the small bowel. If from one-quarter to one-third the total solids of the healthy faeces is composed of bacteria, what is equally striking is that these are nearly all dead. It is an utter fallacy to picture the contents of the colon, as I fancy most of us are apt to do, as undergoing acute putrefaction. Such putrefaction, when it occurs, takes place mainly in the ileum. It is in the small intestine that there is the greatest bacterial proliferation and activity, and there, and not in the colon, we should locate the main absorption of bacterial poisons. Added to this, we have clear evidence that the mucosa of the colon is a region of active excretion; we know, for example, that antimony and mercury are discharged from the blood by this path, while the recent studies of Hertz [3] demonstrate that the ileo-caecal valve does not so much function as a means of preventing regurgitation from the caecum into the ileum as, on the contrary, of retaining the fluid chyme in the ileum until, with the taking of food into the stomach, there is a reflex opening of the gate and filling of the caecum, accompanied by peristaltic propulsion of the contents of the upper colon into the sigmoid. As Hertz had previously pointed out, this filling of the sigmoid has a siphon-like action; when the lower limb of the loop becomes full the whole column descends into the rectum, which normally, until this event, has been empty, and it is the pressure of this column of faecal matter upon the pre-anal portion of the rectum that initiates the desire for evacuation or call to evacuate.

*NOTE.—While saying this I have to confess that I am not wholly convinced that a low form of infection may not play a part in the initiation of these bands. The studies of my colleague, Dr. Archibald—studies extended over a long period, and not yet published, which he permits me to refer to—upon the production and arrest of adhesions in the abdomen of the dog, appear to teach that however careful the laparotomy, bacteria are constantly to be found in early adhesions; that bacteria pass from the intestine into the peritoneal cavity with comparative ease, and that where present locally in any number, and of low virulence, they tend to be destroyed by the inflammation which they set up, so that the fully formed adhesions are sterile. Probably, therefore, such bacteria incite the first formation of plastic adhesions locally, but later, just as in the repair of bone we find the early cellular and inflammatory callus becoming the matrix in which laminae of true bone are laid down along the lines of stress, so here, the plastic exudate affords the matrix in which, once laid down, the connective tissue growth may progressively increase along the lines of stress in harmony with the extent of that stress.

As McCrae and I have pointed out elsewhere,[9] the very arrangement and relationships of the appendix, caecum, and colon indicate function. Now, it is quite true that, as with all other organs of the body, there is in the alimentary canal a huge reserve force and margin of safety; but because it has been demonstrated that very little absorption takes place from the stomach, and that a man can manage to exist without a stomach, that is no reason to recommend gastrectomy as a popular proceeding, as the proper course to pursue in chronic dyspepsia, for example, however excellent a measure this might be in cancerous states or obstinate ulceration. We shall soon at this rate have it recommended that, as after the climacteric the breasts and uterus are of no possible use, and as more than any other organs they are liable to be the seat of cancer, therefore as a matter of routine these organs be removed between the ages of 40 and 50.

Saying this I do not wish it to be understood that (leaving out of consideration malignant growths) I recognize no conditions in which ileo-sigmoidostomy is to be recommended. I freely admit that the mechanical effect of retained faeces may induce congestion and inflammation of the mucosa of the colon with altered secretions favouring the growth of bacteria, which in their turn induce a general degeneration of the mucosa with erosion and ulceration of the viscus, and that this colitis, if long continued, may so alter the mucous membrane that normal function cannot be restored. In such cases the one hope of giving back relative health to the patient lies in colectomy. But, with Hale White,[10] I would say that it casts a grave reflection upon the medical attendant if a patient be permitted to reach this state. There are more means than one of promoting the action of the large bowel—more means than saline purgatives and vegetable cathartics; more means than Sir Arbuthnot Lane's soft paraffin and mechanical support of the lower abdomen—in fact, I am inclined to think that such passive support is distinctly an erroneous treatment. If my colleague, Professor Geddes, be right, and laxity of the abdominal wall be the prime cause of displacement of the bowels with consequent stasis, then active increase in the muscular tonus, and not passive bolstering up from without, is what is indicated—in short, a course of abdominal massage. Not being in practice, I speak with some diffidence about these matters of treatment. I recall, however, as from the distant past, that this laxity of the abdominal wall was what I was taught as a main cause of chronic constipation in my student days, and that my old chief, Dr. Morgan of Manchester, in the Eighties used to recommend for those who could not afford the services of a professional masseur, that they should beg, borrow, or steal a small cannon ball and roll it morning and evening for so many minutes over their abdomen. He explained that its very coldness had a tonic effect, and that rolling up the right side and down the left might also duly stimulate the colon to contraction.

In short, while granting freely that Sir Arbuthnot Lane in certain cases has by the removal of a permanently damaged colon given a new lease of life to his patients, converting chronic and progressive invalidism into health and activity, I must at the same time accuse him of false doctrine in his teaching that the colon is merely a "common sink." The danger of this false doctrine is that, encouraged by Sir Arbuthnot Lane's brilliant advocacy, encouraged to regard the colon as of no account, the immature surgeons of two continents will inaugurate an era of short-circuiting, performing this or the yet graver colectomy for all sorts and conditions of disease in all sorts and conditions of men, women, and children, on the smallest possible pretext. It is, I hold, too grave a responsibility to assume, this of starting an epidemic of operative surgery purely upon an empirical basis, on a foundation that is not established upon uncontrovertible fact. For, to repeat, those who employ this term "autointoxication" do not know what they are talking about; they do not know whether the morbid state originates in the small or the large bowel; they see at most that delayed passage in any part of the alimentary tract leads to serious symptoms, but whereas if the hand had offended them they would not cut it off until they had employed the latest and last efforts of conservative surgery, with the colon they would have no such scruples.

THE POSITIVE DATA CONCERNING INTESTINAL INTOXICATION.

This brings me to the second question referred to some time ago, namely, the nature of the processes set up by intestinal stasis. The truth is that we know painfully little that is precise. Take first the matter of intestinal intoxication—of absorption, that is, of deleterious materials developed in the course of intestinal activity. There are, it would seem, three orders of possible toxic substances to be taken into consideration: (1) The products of disintegration of foodstuffs by the digestive juices, (2) the products of disintegration of foodstuffs by bacterial activity, and (3) the ectotoxins discharged by the intestinal bacteria.

As to the first we know this, that the gastric juice splits the proteins into the simpler diffusible peptones and proteoses, the pancreatic juice can act on both proteins and peptones, the succus entericus cannot act on proteins, but can act on peptones and proteoses, splitting them into bodies of the order of polypeptids and amino-acids. Now peptones, and more especially primary proteoses introduced directly into the tissues, are directly toxic.[11] The studies upon enteral and parenteral digestion respectively have, however, demonstrated that normally in the passage through the intestinal wall these proteoses are either broken down or built up into harmless substances; it is when they gain entrance directly into the tissues that they are dangerous. Nor can it be urged that where the intestine is inflamed or ulcerated, bodies of this order gain entrance into the blood and lymph, and so set up the symptoms encountered in chronic constipation, because the symptoms of peptone and proteose intoxication are of a totally different type, resembling in fact those seen in anaphylactic shock. While laying this down I would point out that there is still lacking any thorough study upon the effects of entry into the system of recurrent minimal doses of bodies of this order. But for the present they cannot be implicated.

It is not a little interesting that as the protein molecule undergoes further disintegration into bodies of the nature of polypeptids and amino-acids, the products, in general, instead of becoming more toxic, become less toxic. There are some exceptions which I shall note shortly, but so far as I can gather, digestion by the gastric and intestinal juices pure and simple, conducted *in vitro*, affords no substance to which we can ascribe any important group of Sir Arbuthnot Lane's series of symptoms.

In like manner the products of carbohydrate and fatty disintegration have so far not been found to yield disturbances resembling any considerable group of Lane's symptoms, or, to my knowledge, even one of the seventeen, unless we here include the wasting that occurs from lack of digestion and absorption of fatty and other foodstuffs.

We must conclude, therefore, that in the light of our present knowledge, it is not the digestive fluids that by their action on the foodstuffs induce Lane's symptoms.

THE PRODUCTS OF BACTERIAL ACTIVITY UPON THE FOODSTUFFS.

With the products of the fermentative and disintegrative activities of the intestinal bacteria the case is somewhat different, but still vague and unsatisfactory. It is well determined that the different bacteria acting upon soluble foodstuffs give rise to different dissociation products, but it cannot be said that the testing of these products, steadily pursued as it has been for the last quarter of a century, has led us very far. Some here may remember the excitement that was raised by Brieger and his ptomaines in the years that immediately followed Bouchard's lectures. To-day we rarely hear the word, the conclusion having been reached that these diamins, results of bacterial activity—for example, putrescin and cadaverin—are produced in such relatively small amounts that they may be neglected. At most there is still some talk regarding one of these possible final products of protein degeneration (but more frequently of lecithin dissociation), namely, cholin and its conversion into its more poisonous ally, neurin. It cannot, however, be said that this has progressed beyond the stage of hypothesis; or that, from what we know of the action of neurin, it could be made responsible for more than certain of the nervous and possibly of the muscular disturbances.

Our only positive data are in connexion with another series of disintegration products, the results of the disintegration of the aromatic group in the protein molecule—tryptophane and its derivatives, indol, skatol, phenol, and cresol. We owe to that devoted physician and pathological chemist of New York, the late Dr. Herter,[12] the determination that indol introduced into the circulation induces some of the striking symptoms which we associate with retention of faeces—headache, mental irritability, muscular fatigue. Although this has not been proved, Vaughan Harley[13] and others ascribe the melancholia and neurasthenia of chronic constipation to excessive continued absorption of indol, skatol, and phenol from the bowel.

But, granting that in indol and its allies we have discovered the cause of the predominant set of symptoms in simple constipation, we are met with the following contradictory facts—namely, that indol and skatol are not easily absorbed from the large intestine. Time and again it is possible to obtain abundant indol from the stools when the urine affords scarce a trace of indican: dry scybala, for instance, are often rich in unabsorbed indol. It is now well established that, judged by the amount of indican in the urine, it is when obstruction occurs high up in the small intestine with retention and putrefaction of the fluid faeces in that region, that the most indol is absorbed and excreted through the kidneys. It is quite possible that when the fluid contents of the ileum pass into the caecum, and there by resorption of the fluid undergo desiccation, some indol is taken into the system, but this is not the main source.

Evidently, therefore, if these symptoms are due to the indol group, the colon only plays, in general, a secondary part in their development. We must suppose that a full colon causes retention of fluid faeces in the ileum, and so favours absorption of the indol group, or suggest that while little absorption occurs in the healthy colon, the inflamed mucosa takes up the indol and its allies. Once again we are led to the conclusion that symptoms are directly ascribed to the poor colon which belong elsewhere. I cannot but feel that there is considerable truth in a remark made to me within the last week by Dr. Harvey Cushing: "In seeking to gain a knowledge of the disorders of the alimentary tract we are utilizing the methods of the tunnel borer, and have started at both ends; we have passed now through the stomach to the duodenum at one end, through the rectum to the ileo-caecal valve at the other; we ascribe every digestive disturbance to these parts about which we know something, but in the meantime the whole length of the small intestine is absolutely unexplored; to it we ascribe nothing."

Evidently we can proceed very little further; my old friend, Professor Woolley of Cincinnati, has been studying the effects of long-continued injections of indol and tyrosin. He and Newburgh[14] have by this means been unable to discover any changes in the glandular organs; no blood vessel changes were observed in any case either in the indol or the tyrosin series. There was a slight effect upon the adrenals—namely, an increase in the pigmentation of the chromaffin cells of the medulla together with a relative increase in the size of the cortex. But this was all.

Interesting as it is, the work of Ackermann,[15] Mellanby and Twort,[16] and others, upon the production from histidin of toxic β imidazolylethylamin by putrefactive and intestinal bacteria, cannot as yet be surely correlated with the symptoms due to intestinal stasis; indeed, as bearing upon the difference between enteral and parenteral digestion, we have the observations of Barger and Dale[17] that "β i." is easily obtainable from the fresh mucosa of the small intestine removed during life, and this simply by treatment with alcohol, so that highly toxic as it is there is evidently a mechanism whereby this substance does not gain entrance as such into the general circulation: at most it may be responsible for the acute death which is apt to follow upon complete duodenal obstruction. As a matter of fact, this work of Herter, and its continuation by Woolley and Newburgh represents the one solid acquirement we have gained in this matter of alimentary intoxication, and you see how doubtfully it can be applied to the colon and to the development of the more serious states which may accompany grave coprostasis.

BACTERIAL ECTOTOXINS.

We come now to the third form of intoxication—that, namely, by bacterial ectotoxins. It is striking that the intestinal bacilli *par excellence*—namely, the members of the *B. coli* group—produce no recognizable ectotoxins; the same is true of the other important group of the streptococci. If either of these directly caused the symptoms of alimentary intoxication, it could only be through bacteriolysis, through breaking down and liberation of their split products. Now, in the first place, accepting Vaughan's most thorough work,[11] these bacterial split proteins cause symptoms similar to those set up by the peptones and proteoses, not those of faecal retention; and in the second, the vast number of bacterial corpses seen in the faeces is contrary to any theory of bacteriolysis; the bacteria have been killed, but have not suffered dissociation and dissolution.*

There are only two recognized invaders of the alimentary tract that produce ectotoxins known to produce intoxicative symptoms when absorbed. These are the rare *B., botulinus*, which by its toxins acts particularly in the nervous system, setting up one form of meat poisoning, and the *B. pyocyaneus*, a not uncommon inhabitant of the ileum and lower bowel, whose toxins act also in the nervous system. So far I think I am right in saying that whenever the characteristic tonic contraction of the muscles have been observed, which are brought about by *pyocyaneus* toxins, we have had to deal, not with a mere intoxication, but with a definite infection by this organism. That, at least, has been our experience in Montreal. There still is needed a careful study of the anaërobes of the digestive tract, which, as Herter pointed out, are apt to be greatly increased in irritative conditions of the lower bowel. By analogy some of these should produce powerful ectotoxins. It does not, however, follow that such ectotoxins, if discharged, undergo absorption by the bowel wall.

Here once again it is evident that there is singularly little evidence of the development of intoxication proper.

SUBINFECTION VERSUS INTOXICATION.

When, indeed, we sum up all these known data, we find that they are so mean in amount that irresistibly I find myself inclined to ask, "Are we nosing along the right scent? May not we be working along the wrong one?" Are there not observations of another order that throw much more light upon Lane's syndrome with its associated diseases, and at the same time harmonize other observations regarding the onset of the same group of diseases—an onset by no means necessarily related to intestinal stasis?

If I may criticize Sir Arbuthnot Lane's reports upon his cases, I would say that these are not up to date—they are too largely clinical. An important development nowadays, such as he recommends, demands the fullest co-ordination with the laboratory; there ought to be routine blood cultures in every case recorded, routine examination and reports upon the stools and their predominant bacterial types, blood counts, haemoglobin examinations—in fact, the full clinical study of each case, so that nothing is neglected. That, I say, is demanded to-day; that Sir Arbuthnot does not give us. The surgeon to-day, no less than the physician, must so arrange his service as to give his patients all these aids to correct diagnosis. What interests us, however, is to note that occasionally in his series of cases Sir Arbuthnot notes that either *B. coli* or streptococci, or both, were found upon blood culture. The point I want to make is this—that had Sir Arbuthnot instituted fuller preliminary studies he would have recorded this finding in so many of his cases that he would not have spoken of autointoxication, but of low infection, as the cause of most of the disturbances he tabulates.

Fourteen years ago I called attention to this matter, bringing forward a considerable body of evidence.[18] I showed, both from the studies of others and the investigations of workers in my laboratory, of Ford, Nicholls, and others, that not only do the lymph nodes of the respiratory tract and of the alimentary tract of normal animals constantly afford cultures of bacteria, but also that properly prepared organs, such as the liver and kidneys of healthy animals, yield cultures of pathogenic and non-pathogenic bacteria; that, through the agency of leucocytes, bacteria are constantly being carried into the system and as constantly being destroyed in the healthy animal; that with inflammatory conditions in the alimentary canal and greater accumulation of leucocytes in its walls there must be greater passage of these from the surface and more extensive carriage of bacteria into the system. According to the virulence and number of these bacteria so may there be set up other foci of active infection or a condition which I termed "subinfection"—that is to say, the bacteria thus carried in do not multiply and set up foci of suppuration; they are destroyed, but with their destruction the liberation of their toxins causes a poisoning of the cells immediately around them, and the accumulative action of these toxins, whether locally or at a distance, upon the liver cells, for example, brings about the death of certain cells and replacement by fibrous tissue.

In the fourteen years that have elapsed since I brought forward these ideas upon subinfection steadily and surely facts have accumulated in support of these views and the allied view regarding the frequency of latent infection. There has been abundant confirmatory evidence demonstrating that tubercle and other bacilli fed by the mouth to young animals are to be found in the lymph of the thoracic duct in the course of an hour or two. We now know that whether the point of entrance be through the upper respiratory tracts or through the digestive tract, by the eighteenth year 95 per cent. of those examined respond to the tuberculo-cutaneous tests, although only 10 per cent. die from the disease; in 85 per cent. of the population the disease becomes arrested and latent. Dr. Opie[19] has demonstrated that portal cirrhosis is a subinfection by showing that whereas chloroform alone will not cause cirrhosis in the animals of the laboratory, although it causes necrosis of the liver cells, which subsequently regenerate, and whereas inoculation of sublethal doses of colon bacilli alone will not cause cirrhosis, chloroform, followed by sublethal doses of *B. coli*, surely will. I will not go into these matters here; a year ago I referred to them in Iowa City.[20] What I want to point out is that properly made blood cultures afford a high proportion of positive results in cases of continued intestinal stasis, and that most, if not all, of Lane's symptoms which have not so far been demonstrated to be due to intoxication are such as follow subinfection, not necessarily originating through the lower bowel, but due to the carriage in of bacteria through the mucous membrane at any point from the mouth to the anus.

I will not say—that would be absurd—that intoxication by the indol group, for example, plays no part. I would only say that our present knowledge of the phenomena of enteral and parenteral digestion suggests that the toxic products of protein (including bacterial) disintegration are most effective upon the tissues when they are produced within them, and not upon the outer side of the mucous membrane. Thus, to conclude by recapitulating Lane's symptoms:

Loss of fat (1) and wasting of the muscles (2) can be induced by inoculating animals with recurrent sublethal doses of *B. coli*. Such recurrent inoculation of sublethal doses of *B. coli* or of streptococci induces haemolysis and liberation of blood pigment in the circulation(3). As shown some years ago by Charlton[21] in my laboratory, a grave condition of anaemia may be induced by this means in laboratory animals. The pigmentation reaches an extreme degree in "microbic cyanosis," the condition first described by Stokvis, in which the late Dr. Gibson of Edinburgh,[22] and Blackader and Duval of Montreal[23] have found as obvious cause an acute *B. coli* bacteriaemia. The papules and pimples of the skin gain their simplest explanation as being of the same nature as the rose spots in typhoid fever, that is, as brought about by lodgement of bacteria in the cutaneous capillaries.

The offensive perspiration I must leave an open matter. I am quite willing to accept it as due to the diffusion of volatile bodies of the skatol type from the intestinal tract.

Subnormal temperature (4) is one of the marked features of *B. coli* infection. As regards the symptoms of mental irritation or torpor, and the melancholia (5), as already

*Here is another piece of work that deserves doing—namely, to observe whether in prolonged retention of faeces the faecal bacteria exhibit any evidences of lysis.

stated, I freely accept these as set up by members of the indol group, leaving it an open question whether these be of enteral or parenteral production. It deserves note, however, that *B. coli* inoculations also affect the nervous system, the most striking result being the production of paretic changes of an ascending type.

Chronic Interstitial Fibroses.

Regarding the rheumatic aches and pains (6) and their origin it is necessary to speak a little more at length, and that because only within the last few weeks rheumatic myositis has been for the first time experimentally induced and given an adequate explanation. These experiments throw a flood of light upon a most important group of so-called chronic rheumatic or rheumatoid lesions. It was my good fortune upon my journey here to spend some hours in Chicago with Dr. Rosenow, and there to study his exquisite material bearing upon the development of various strains of the streptococcus and the lesions produced by these different strains.[24] Among these were the lesions of myositis in the rabbit. These lesions he is able to produce with certainty, and in the very regions in the rabbit in which in man the rheumatic aches and pains show themselves. They are not of toxic origin, but of the nature of a typical subinfection. Studying the lesions at various periods of their development it is seen that streptococci of a particular grade of attenuation become arrested in the muscle capillaries, more especially in those situated near the tendinous attachments. For a few hours it appears that these multiply; following upon this a small area of muscle fibres in the immediate neighbourhood of the affected capillary undergo necrosis and become pale. And here is the striking feature—the cocci do not proliferate further, but, on the contrary, disappear, undergoing lysis. There is no formation of a productive microbic lesion, no formation of an abscess for example. It appears that so soon as the bacteria set up a reaction, whether by their mere mechanical presence, or by endothelial phagocytosis of some of their number and liberation of their endotoxins, that reaction is adequate to destroy the rest of the bacteria, and this with relatively very slight accumulation of wandering cells. But the liberated and diffused endotoxins are sufficiently powerful to destroy the more highly differentiated cells in the immediate neighbourhood, and following this destruction there is either, in favourable cases, regeneration and *restitutio ad integrum*, or, in unfavourable cases, a replacement and even a productive fibrosis.

I describe these lesions in some detail because it so happens that only in April last, delivering the Harveian Lecture before the Harveian Society of London, Dr. Luff[25] very serviceably brought together all the examples of these chronic "rheumatic" affections, pointing out that the lesion is of the nature of a hyperplasia of the ordinary connective tissue in various points of the body, that the condition may undergo absorption, or may pass on to organization with the formation of nodules or patches of thickening. The parts implicated are the fibrous tissues of the joints, muscles and bones, aponeuroses and insertions of muscles, "the sheaths in which the muscle spindles lie," the bursae, fasciae, ligaments, and capsules of joints, and the periosteum. The indurations may be widespread, but generally are well defined, and vary in size from ½ in. to 1 in. in diameter.

Luff would group all these lesions under the comprehensive term "fibrositis." I will not here discuss whether this term is to be accepted. What I would point out is that he describes lesions identical with those produced experimentally by Rosenow. It is quite true, as he points out, that these most often occur without any previous attack of acute rheumatism. That, however, does not mean that they have not a similar etiology. As Rosenow has, I think, conclusively demonstrated, the reason why acute rheumatic endocarditis, for example, originates only in youth is purely anatomical. The typical verrucose endocarditis of acute rheumatism is of embolic origin, and can be induced by inoculating half-grown rabbits with streptococci of a grade of virulence closely allied to that which in older rabbits will induce the above-mentioned myositis. Why the older animals do not suffer from endocarditis is because in them the heart valves have become non-vascular, the fine arterioles present in early life in the proximal two-thirds of the cusps undergoing obliteration. In all probability similar vascular changes explain the incidence and location of rheumatoid changes at different life-periods.

Following the older tradition, Luff regards this fibrositis as of toxic origin, although he gives no experimental evidence in support of this view, and interestingly enough quotes Ware as having demonstrated the presence of gonococci in the myositis secondary to gonorrhoeal arthritis. Here I would point out what Luff neglects to notice, that at times "muscular rheumatism" accompanies acute rheumatic arthritis of the ordinary non-gonorrhoeal type. By analogy I would suggest that the muscular aches and pains of influenza are due to similar embolic lodgement and lysis of the influenza bacilli in the muscle capillaries.

More and more, in fact, are we coming to realize that various orders of chronic interstitial fibrosis originate from this condition of subinfection. I have already referred to Opie's experimental production of hepatic cirrhosis by action of the *B. coli* in the damaged liver. Recently Gaskell,[26] working under Aschoff, and Libman and Baehr[27] in New York have drawn attention to a form of chronic Bright's disease set up by streptococcal embolism in the fine capillary loops of the glomeruli accompanying subacute endocarditis of streptococcal origin. In all these cases, let me emphasize, we have at most a temporary proliferation of bacteria in the capillaries to which they have been carried, until such time as mechanically or otherwise they set up irritation and reaction; so soon as the tissues react the bacteria undergo lysis. It is not their proliferation but their death that liberates the toxic substances, which, diffusing out, destroy the higher tissue cells in the neighbourhood, and simultaneously stimulate the lower connective tissue cells to proliferate and develop an area of fibrosis. We deal, that is, with something beyond local capillary infarct formation; the area of necrosis extends beyond the limit of the territory of the blocked capillary; its extent can only be ascribed to the action of liberated toxins. What is characteristic of all these cases is recurrence, or, more accurately, they represent not simultaneous infection, but the summation of a succession of minute insults to the tissues, sometimes occurring within a few days or weeks, but often extending over years, so that on examination lesions may be discoverable in various stages. What is more, it is evident that the incriminated bacteria gain this recurrent entry into the blood stream from various points—from the genital passages in gonorrhoea, from the tonsils in rheumatic tonsillitis, from the gums in pyorrhoea alveolaris, and it may well be from the intestines in cirrhosis and many other conditions. With this light upon the nature of the process, it is now our duty to study the bacteriology of each case, and from that determine the treatment.

Having said this much, I can be very brief regarding the remainder of Sir Arbuthnot Lane's symptoms and diseases. The arterial, renal, and eye changes (Nos. 12, 13, and 17) belong to the "fibrositis" group; the pancreatic and hepatic changes (Nos. 15 and 16) have all the ear-marks of the like subinfection. The very admission that the blood pressure is at times increased, at times lowered (No. 8), is indication that we deal with no one common basic intoxication; it is what might be expected in subinfection by different orders of bacteria. But this and the atrophy of the breasts (No. 9) must properly be left as open matters. Lastly, as regards the atrophy of the thyroid (No. 7), it deserves note that the work of the last year points to this as a result of infection. Farrant[28] has shown that diphtheria and other pathogenic bacteria inoculated into experimental animals induce excessive activity of the thyroid, and indicate that an important function of the gland is by its internal secretion to neutralize circulating toxins. Continued stimulation of the gland must result either in exhaustion and atrophy or, as Lane points out, in adenomatous overgrowth.

To those who have not been following the drift of recent bacteriological research, this latter part of my address may appear to be of the nature of special pleading. Be it so. I shall be perfectly satisfied if it leads my clinical brethren to examine more carefully into these cases of chronic intestinal and rheumatoid cases, of cirrhosis and Bright's disease, in fact, of all the conditions commented upon by Lane, to determine whether evidences of infection

or subinfection are to be detected. I would, however, in all meekness, object that the intoxication theory has been tried, and, save as regards a singularly limited series of symptoms, has so far been found wanting when subjected to the test of experiment; while, on the contrary, it is possible to adduce experimental evidence in favour of each of my pleas in favour of the infective nature of the conditions discussed.

CONCLUSIONS.

To epitomize:

1. It is more rational to regard the evil effects of intestinal stasis as, in the main, a result of conditions favouring subinfection and low forms of infection than as a result of chronic intoxication.

2. The term "gastro-intestinal autointoxication" is pernicious and not to be employed by any self-respecting member of our profession, save for so limited a set of conditions that for ordinary purposes it may safely be wholly expunged from the medical vocabulary.

3. While the symptoms and diseases enumerated by Sir Arbuthnot Lane may follow intestinal stasis, at least a large proportion of them may originate independently of such stasis.

4. Before recommending the operation of short-circuiting it is necessary, therefore, to make the fullest studies, so as to discover, if possible, the nature of the organism responsible for the disturbance and its probable seat of entry.

5. A discovery of the cause of the symptoms is calculated to suggest the appropriate means of treatment by means other than short-circuiting. Only when these have been tried and found wanting is removal or short-circuiting of the colon justifiable.

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UNDER the will of the late Mrs. Sarah Berry of Wimbledon the Royal Hospital for Incurables at Putney receives a bequest of £1,000.

IN a pamphlet entitled, *What Town Planning Means*, published by the Garden Cities and Town Planning Association (post free, 3d.), the author, Mr. George Pepler, F.S.I., in addition to discussing what local councils and private owners of property may do under the Act, and the possibilities that the Act opens up, gives a list of corporations and urban district councils which have already prepared, or are considering the preparation of, schemes of improvement. They number not far short of a hundred. In a good many localities schemes for dealing with several different areas are in view.

An Address
ON
CHRONIC INTESTINAL STASIS.

DELIVERED BEFORE THE DUBLIN UNIVERSITY BIOLOGICAL ASSOCIATION ON NOVEMBER 22ND, 1913.

BY SETON S. PRINGLE, F.R.C.S.I.,

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ALL of us have had the experience of treating and some of us of operating on cases with more or less indefinite abdominal symptoms—symptoms which may have suggested gastric or duodenal ulcer, gall stones, chronic appendicitis, ovaritis, etc. But at the operation none of these lesions have been found, or if found, their appropriate treatment has failed to permanently relieve our patients. Looking back over such cases we recognize that in many the abdominal trouble has been accompanied by general debility, chronic constipation, a sallowness or duskiness of the skin, headaches, bad appetite. In fact, the patients were toxaemic, and in treating the lesion we have merely dealt with the result and left the cause untouched. The root of the trouble in these cases is chronic intestinal stasis, or, in other words, delay in the passage of the contents through the alimentary tract, and consequent excessive absorption of toxic products. We are indebted chiefly to Sir Arbuthnot Lane for enlightenment on this subject, and no communication such as this would be complete without heartily acknowledging the excellent pioneer work he has accomplished in the face of much prejudice and keen opposition. Many other workers have thrown light on one or other aspect of the problem, and I wish to mention in this connexion the names of Coffey, Wilms, Flint, Mayo, Jackson, Connell, Rovsing, Goldthwait, Bainbridge, Eastman—and last, but not least, Mr. Gray. Personally, I have been feeling my way in the treatment of these most interesting cases for the last five years. As my own ideas have become crystallized, I have absorbed and adopted many of the ideas put forward in the first place by some of the above writers, so here and now I wish to express my obligation.

There are, to my mind, two distinct factors in the production of stasis: (1) Ptosis of some part of the gastro-intestinal tract, and (2) kinking and stenosis of the bowel by bands and membranes; in other words, we may speak of "stasis of gravity" and "stasis of obstruction." These two conditions are as a rule associated; but in such cases one condition generally preponderates and overshadows the other.

ANATOMICAL CONSIDERATIONS.

In the study of ptosis the first matter to consider is the manner in which the various parts of the gastro-intestinal tract are maintained in position against the force of gravity which is constantly tending to produce downward displacement in the erect position assumed by man. If we glance at the longitudinal section of the abdomen we are at once struck by the fact that the shape of the abdominal cavity proper, as distinct from the pelvis, is that of an inverted cone. It will also be seen that the posterior abdominal wall forms a shelf inclined at an angle of about 50 degrees, on which all the heavy organs rest. The majority of the organs are supported on this shelf by peritoneal folds and mesenteries, by pads of fat, and by the abdominal pressure maintained by the anterior abdominal wall. In the case of the duodenum and the ascending and descending colon we also have direct attachments to the fascia of the posterior abdominal wall—a state of things brought about by the fusion and disappearance of the peritoneal coverings of the gut and parietes where these come in contact. If one of these means of support is lessened, equilibrium is disturbed and ptosis results. Thus ptosis is in the early stages localized, the commonest condition being either mid-line ptosis where the stomach and transverse colon are affected, or right-sided ptosis where the caecum is the organ at fault. In many cases the condition does not progress further; but on the other hand, if stasis is caused by the ptosis, we get distension behind the affected part, with general ill-health and resultant absorption of fat and loss of muscle power.